diseases'' of the heart. In about 20–30% of patients with Brugada syndrome, genetic mutations of the α subunit of the cardiac sodium channel (SCN5A) can be identified. Assessment of these mutations in expression systems demonstrated loss of function of the sodium channel (in contrast to the gain of function of the sodium channel in LQT3). The electrocardiographic manifestations of Brugada syndrome may be transient or concealed but can be unmasked or challenged with sodium channel blockers (ajmaline, flecainide, and others), vagotonic stimulation or fever. The diagnostic and prognostic impact of an incidental finding of Brugada-type ECG signs in asymptomatic individuals without a family history represents a controversial and currently unresolved, yet growing problem in clinical decision making. Currently, ICD implantation is the treatment of choice in secondary and primary prevention of sudden death in high risk patients with Brugada syndrome.[20]

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In compliance with EBAC/EACCME guidelines, all authors participating in Education in *Heart* have disclosed potential conflicts of interest that might cause a bias in the article

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